

Clinical Profiles of Lateral Medullary Syndromes in a Tertiary Care Center in Chengalpattu District

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ABSTRACT

Objectives: To describe the clinical presentation, risk factor profile, imaging findings, and outcome in patients diagnosed with Lateral Medullary Syndrome (LMS) in a tertiary care center – Chengalpattu district.

Methods: A hospital-based cross-sectional study was conducted on 11 patients diagnosed with LMS presented to General Medicine OPD in Karpaga Vinayaga Institute Of Medical Sciences, Chengalpattu district between January 2023 and January 2025. All patients underwent detailed clinical evaluation, neuroimaging (MRI with MRA), and assessment of vascular risk factors.

Results: Out of 11 patients, 9 were male and 2 were female, with a mean age of 56.3 years. The most common risk factor was diabetes mellitus for 7 patients followed by dyslipidemia for 6 patients and hypertension for 5 patients. All patients presented with limb ataxia and vertigo. Other common findings included nausea and vomiting for 9 patients, dysphagia for 5 patients, hoarseness of voice for 4 patients, hiccups for 4 patients and Horner's syndrome for 6 patients. MRI confirmed dorsolateral medullary infarction in all cases. MRA revealed vertebral artery involvement for 5 patients and Posterior inferior cerebellar artery for 6 patients.

Conclusion: LMS in our cohort predominantly affected older males and was strongly associated with vascular risk factors such as diabetes and dyslipidemia. Ipsilateral limb ataxia and vertigo were consistent findings. Imaging plays a vital role in diagnosis and identifying underlying vascular pathology.

Keywords: Lateral medullary syndrome (LMS), Wallenberg syndrome, posterior circulation stroke, diabetes, vertebral artery, Posterior inferior cerebellar artery.

INTRODUCTION

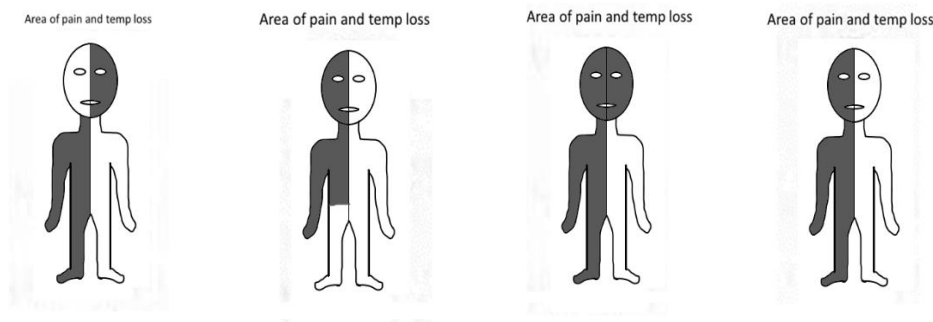
Lateral Medullary Syndrome (LMS) also known as Wallenberg's syndrome, is a classical example of a posterior circulation stroke affecting the lateral aspect of the medulla oblongata. The condition is most frequently caused by occlusion of the posterior inferior cerebellar artery (PICA) or the vertebral artery. The underlying mechanisms are often related to large artery atherosclerosis, cardioembolic events, or arterial dissection, particularly in younger patients [1,2]. Owing to the compact arrangement of critical neural structures in the lateral medulla, even a small infarct in this area can produce a wide range of distinctive clinical signs.

The clinical profile of LMS reflects injury to multiple neuroanatomical regions, each contributing to specific symptoms. The vestibular nuclei and inferior cerebellar peduncle, when involved, result in vertigo, nystagmus, gait instability, and ipsilateral limb ataxia. Damage to the spinal trigeminal nucleus and tract produces ipsilateral facial hypoesthesia or analgesia, while involvement of the spinothalamic tract leads to contralateral loss of pain and temperature sensation on the body [3–5]. This combination of “crossed” sensory loss is a hallmark feature of LMS.

The nucleus ambiguus, containing motor fibers for the glossopharyngeal (CN IX) and vagus nerves (CN X), contributes to dysphagia, dysphonia, and impaired gag reflex when infarcted. Additionally, disruption of descending sympathetic

fibers results in ipsilateral Horner's syndrome, characterized by ptosis, miosis, and anhidrosis. In some cases, the solitary nucleus may be involved, affecting taste perception and autonomic reflexes, though this is less common [3–5]. From a sensory perspective, the syndrome is most commonly associated with the Stopford Type I pattern, which shows ipsilateral facial and contralateral body sensory deficits. However, sensory variation is not uncommon. The Type IV or pseudo thalamic pattern, for example, presents with contralateral loss of sensation in both face and body, which can easily be mistaken for a thalamic infarct unless brainstem pathology is specifically considered [6,7].

FIGURE 1 - STOPFORD CLASSIFICATION TYPE 1 TO 4 PATTERN OF SENSORY LOSS
TYPE 1(classical) TYPE 2 TYPE 3 TYPE 4



Diffusion-weighted MRI (DWI) is particularly effective in detecting small infarcts in the posterior fossa, while magnetic resonance angiography (MRA) can demonstrate flow-limiting stenoses or occlusions in the vertebrobasilar arterial system [8,9]. Despite these advances, early diagnosis still depends on a high index of clinical suspicion, as many patients may present with partial syndromes, such as isolated vertigo, sensory symptoms, or bulbar palsy without the full classic triad [10].

The importance of early recognition lies in preventing potentially serious complications. Aspiration pneumonia, respiratory compromise, and long-term disabling ataxia are not uncommon in untreated or unrecognized cases. Therefore, clinicians must maintain a strong understanding of the neuroanatomical correlates, vascular territories, and sensory syndromic patterns associated with LMS to ensure accurate localization, prompt intervention, and improved long-term functional outcomes [11].

MATERIALS AND METHODS

Study Design and Setting: A descriptive cross-sectional study was carried out at the Department of General Medicine, Karpaga Vinayaga Institute of Medical Sciences, Chengalpattu, Tamil Nadu.

Study Period: January 2023 to January 2025.

Sample Size: A total of 11 patients diagnosed with LMS based on clinical features and MRI findings were included.

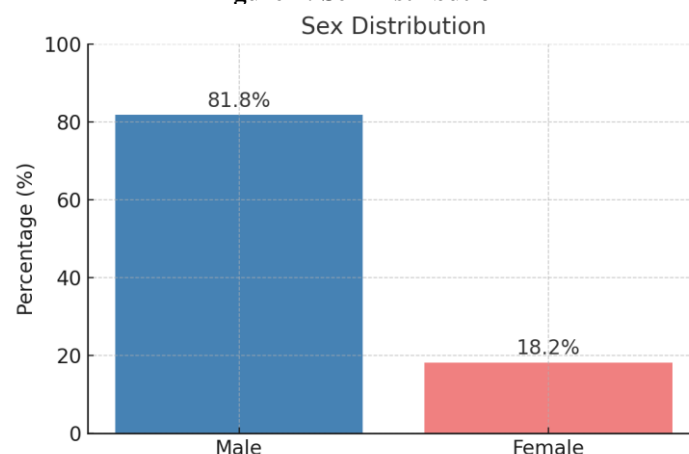
Inclusion Criteria: Adults (>18 years) with acute-onset neurological deficits consistent with LMS and MRI evidence of infarction in the lateral medulla.

Exclusion Criteria: Patients with demyelinating, neoplastic, or infectious etiologies affecting the medulla.

Statistical Analysis: The quantitative data was calculated mean and standard deviation like mean of age is 56.3 years (Range: 44–68 years). Categorical data was presented in charts like bar chart using gender, risk factors and presenting symptoms.

RESULTS:

Figure 1: Sex Distribution



- Mean Age: 56.3 years (Range: 44–68 years)

- Figure 1 demonstrates a strong **male predominance** in LMS cases

FIGURE 2: Risk Factors

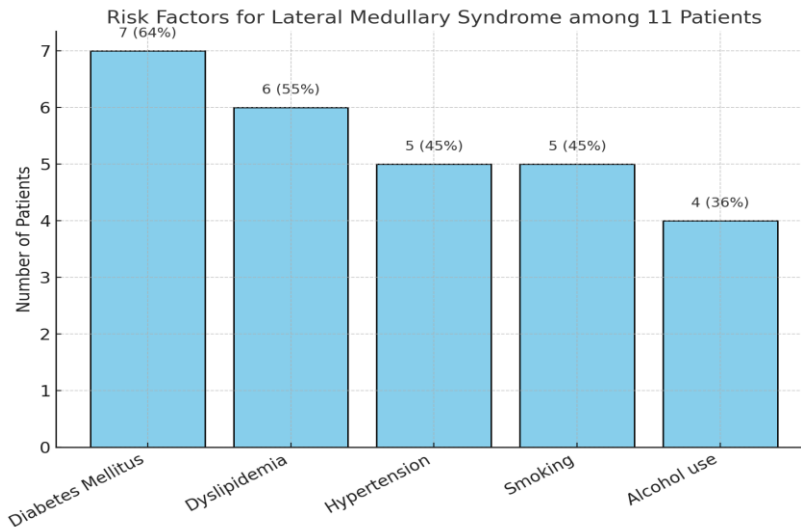


Figure 2 shows the leading risk factor was **diabetes mellitus**, followed closely by dyslipidemia and hypertension.

FIGURE 3: Presenting Symptoms

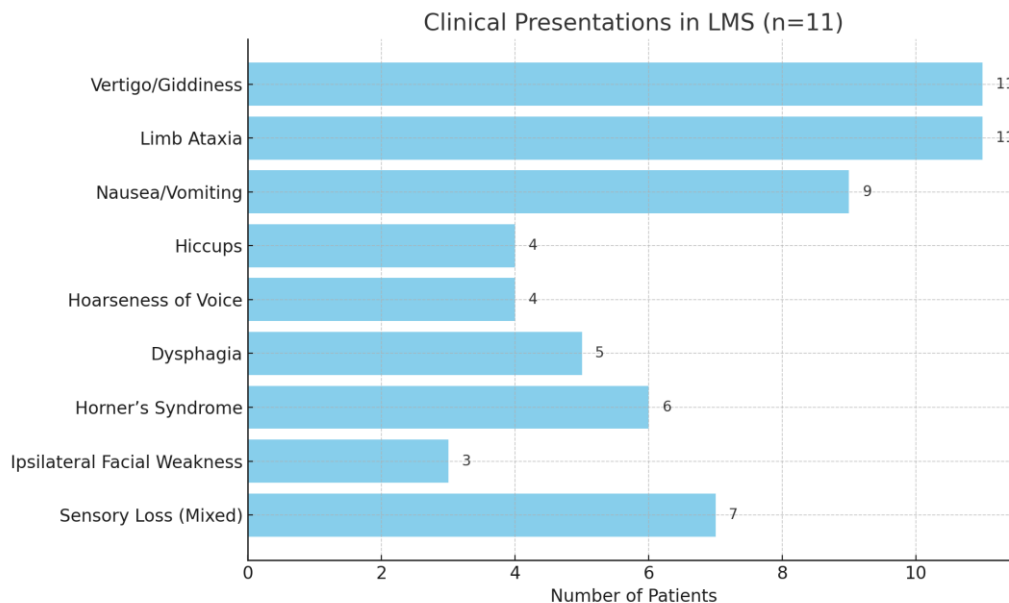
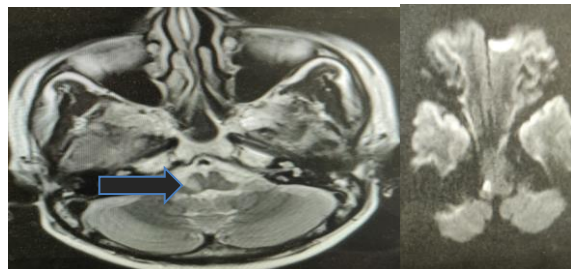


Figure 3 shows all patients exhibited **limb ataxia and vertigo**, reflecting consistent involvement of the **inferior cerebellar peduncle and vestibular nuclei**. Other brainstem-related symptoms like **dysphagia, hoarseness, and Horner's syndrome** were variably present depending on the extent of medullary infarction.

FIGURE 4 (MRI BRAIN – ACUTE INFARCT IN DORSOLATERAL MEDULLA)



Imaging:

- MRI: Acute infarct in dorsolateral medulla (all patients)

- MRA: Vertebral artery stenosis/occlusion (5 Patients) / Posterior inferior cerebellar artery (6 Patients)

DISCUSSION

Our study reinforces the understanding that Lateral Medullary Syndrome (LMS) is predominantly a condition affecting older males, frequently associated with modifiable vascular risk factors such as diabetes mellitus, dyslipidaemia, and hypertension [1,2]. This demographic and risk factor profile is consistent with prior studies by Kim et al. and Amarenco et al., who highlighted the role of atherosclerotic disease in the pathogenesis of LMS [3,4].

Vertigo, gait unsteadiness, and limb ataxia were observed in all patients, reflecting the hallmark cerebellar involvement due to infarction in the inferior cerebellar peduncle or associated pathways [5]. The cranial nerve dysfunction such as dysphagia, dysarthria, and hoarseness typically attributed to nucleus ambiguus involvement [6,7].

Horner's syndrome caused by disruption of the descending sympathetic fibers in the lateral medulla was identified in over half the patients [8].

Neuroimaging, particularly MRI with diffusion-weighted imaging (DWI), was crucial in confirming the diagnosis. Although Magnetic Resonance Angiography (MRA) was unremarkable in most cases, it revealed vertebral artery stenosis or occlusion in 4 patients, highlighting the importance of vascular imaging in the comprehensive evaluation of suspected LMS [9]. Vascular imaging not only aids diagnosis but may also help in prognostication and risk stratification.

One of the most significant observations in our study was the sensory disturbance pattern, specifically Stopford Type IV, characterized by ipsilateral facial and contralateral body hypoalgesia. This pattern correlates with damage to both the spinal trigeminal nucleus and tract (serving the face) and the spinothalamic tract (serving the body) [10]. The quintothalamic tract, a lesser-known but important component, represents the ascending pathway from the spinal nucleus of the trigeminal nerve to the thalamus. Lesions affecting this tract in the lateral medulla leads to ipsilateral facial pain and temperature sensory loss, contributing to the classic dissociated sensory findings seen in LMS [12].

The heterogeneity of clinical presentation in LMS necessitates a high index of suspicion, especially in elderly patients with vascular risk factors presenting with cerebellar or brainstem symptoms. A thorough neurological examination, supported by targeted neuroimaging, remains the cornerstone of accurate diagnosis and timely intervention.

CONCLUSION

Lateral Medullary Syndrome presents with a spectrum of neurological deficits. In this study of 11 patients, it was more common in males above 50 years of age, with diabetes and dyslipidemia being the key vascular risk factors. MRI is crucial for diagnosis and should be complemented with MRA to evaluate underlying vascular pathology. Greater awareness of partial presentations and sensory variants is essential for early identification and management.

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